plete repair should be deferred until the child is 6 to 7 years old to confirm whether the pulmonary valve annulus has sufficiently developed. Various techniques[3,4] for repair must be chosen individually for this subset of anomalies.

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Tricuspid valve in hypoplastic left heart syndrome

To the Editor:

With great interest, we read the article by Ohye and colleagues,[1] describing their institutional experience with surgery for tricuspid valve regurgitation (TR) in patients with hypoplastic left heart syndrome (HLHS). The detailed analysis and clear presentation are praiseworthy. The authors focus on right ventricular (RV) volume overload and annular dilation of the tricuspid valve as the most common mechanism leading to significant TR, whereas structural abnormalities of the tricuspid valve in HLHS are mentioned only briefly as leaflet prolapse and leaflet tethering. We believe, however, that some morphologic features unique to the tricuspid valve in HLHS deserve closer attention.

Some years ago, we specifically studied the tricuspid valve morphology in a series of 82 anatomic specimens of HLHS.[2] One finding was that structural abnormalities of the tricuspid valve were markedly more frequent in hearts with a patent mitral valve. Overall, we found some dysplasia of tricuspid valve leaflets in 35% of the HLHS specimens and in up to 50% of those with mitral stenosis. It would be interesting to know whether the authors' clinical experience also reflects this notion; that is, whether the majority of the patients with HLHS requiring tricuspid valve repair had mitral stenosis rather than atresia. If this were the case, one might infer that patients with HLHS and mitral stenosis are indeed predisposed to clinically relevant TR and might necessitate closer follow-up examinations and perhaps earlier intervention.

Regarding repair of a regurgitant tricuspid valve, the authors favor a partial annuloplasty that effectively eliminates the posterior leaflet and results in a functionally bifoliate valve. Because the coronary artery in the atrioventricular groove can be put at risk with annuloplasty procedures, it is worth noting that a previous study[3] described a significant prevalence of left coronary dominance (56%) in hearts with aortic and mitral atresia compared with regular right coronary dominance in hearts with aortic atresia and mitral stenosis.

The final point we would like to raise is the use of a partial ring for reinforcement of an annuloplasty. In HLHS the septal leaflet of the tricuspid valve differs markedly from that in a normal heart. We found that in hearts with mitral atresia, when the RV aspect of the interventricular septum is concave (ie, bulging toward the left ventricle), the usual direct chordal attachments of the septal leaflet are replaced by additional papillary muscles.[2] The interventricular septum in HLHS becomes essentially part of the RV free wall, and the architecture of the septal leaflet resembles that of the anterosuperior leaflet. Therefore it would be interesting to know how the authors orientate the partial ring, with the opening facing the anterosuperior leaflet or the septal leaflet. With the right atrioventricular valve in HLHS being supported to a greater extent by RV free wall musculature, one wonders whether a closed ring might not be more effective in preventing redilation.

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Reply to the Editor:

I appreciate the comments of Ho and Stamm in their letter to the editor. I am well aware of their excellent article describing the findings of tricuspid valve (TV) abnormalities in 35% to 50% of 82 pathologic specimens of hypoplastic left heart syndrome (HLHS).[1] Because our article was a retrospective, observational review, it did not have the scientific rigor of their study, nor did we have many pathologic specimens available for review. Therefore we concentrated on clinical, rather than morphologic, issues. The only method for evaluation of the anatomy of the TV available to us was echocardiography, which did not always correlate with the intraoperative findings. Because neither of these methods of assessment is particularly robust for describing the exact morphology of the TV, they were not stressed in the article. We also did not attempt to correlate the anatomic subtype of HLHS to the development of tricuspid regurgitation.

We continue to prefer the partial annuloplasty for the repair of the TV in the setting of HLHS, as well as in other lesions. We have not experienced any coronary events or deaths that we thought might be coronary related in patients with HLHS or other anomalies. The few rings we have placed were positioned in the traditional orientation for concerns of the conduction system. Although these patients all universally had excellent results, we have only used them in rare cases in which we thought the relatively fixed annulus would be of sufficient size to carry the child into adulthood.

Thank you again to Drs Ho and Stamm for taking the interest to submit a letter to the editor regarding our article. Such interaction and airing of other viewpoints